Mathematical Theory and Modeling
ISSN 2224-5804 (Paper) ISSN 2225-0522 (Online) DOI: 10.7176/MTM
Vol.9, No.9, 2019

ON ESTIMATING THE CONTROL REPRODUCTION NUMBER OF ZIKA VIRUS DISEASE CONTROL MODEL

Michael C. Anyanwu
Department of Mathematics, Michael Okpara University of Agriculture, Umudike
Email:manyanwu71@yahoo.com
and
Godwin C. Mbah
Department of Mathematics, University of Nigeria Nsukka, Nigeria
Email: godwin.mbah@unn.edu.ng
and
Onwubuya M. Nwabudike
Department of Statistics, Delta State Polytechnic, Oghara, Delta State
Email: mconwubuyanwabudike@gmail.com

Abstract

In this paper, the possibility of stopping the spread of zika virus disease by introducing wolbachia-infected aedes aegypti mosquitoes in the zika endemic area is shown. A system of 14 nonlinear ordinary differential equations is constructed, which models the transmission dynamics of the disease in the human and aedes aegypti populations, including the population of wolbachia-infected aedes aegypti used for control. The expression for the control reproduction number $\mathcal{R}_c$ was derived using the next generation matrix. Numerical evaluation of $\mathcal{R}_c$ at the baseline parameter values shows that $\mathcal{R}_c = 0.1206$ as against the value for the basic reproduction number $\mathcal{R}_0 = 1.123$. This result shows that in the presence of wolbachia-infected mosquitoes, the reproduction number is reduced to below unity, which is the necessary condition for the disease eradication.

KEY WORDS: zika, aedes aegypti, wolbachia-infected mosquitoes, control reproduction number

Mathematics Subject Classification: 92B05, 00A71, 35A24

1 Introduction

Zika virus disease is a mosquito-borne disease caused by a flavivirus. The zika virus is transmitted to humans through the bites of infected female aedes aegypti mosquitoes [9]. Zika virus can equally be transmitted

from man to man through sex, blood transfusion and mother-to-fetus [21].

Zika has been confirmed to cause microcephaly in infants born to mothers who were infected with the virus during pregnancy ([19],[3]). In the recent outbreak of zika in Brazil, there have been over 5000 confirmed cases of microcephaly[9]. In addition to microcephaly, zika has been linked to the neurological disorder, Guillain-Barre Syndrome(GBS) [1]. These associations of zika with these health conditions, prompted the WHO to declare zika virus disease a Public Health Emergency of International Concern in February 2015 [29]. Zika virus disease was first reported in Nigeria in 1954, during investigation in Afikpo, Eastern Nigeria, of an outbreak of jaundice suspected of being yellow fever, [16]. In 2007, zika outbreak occurred in Yap Island, Federated state of Micronesia, in the North pacific ([12], [7]). There was a severe outbreak of zika virus disease in French Polynesia between 2013 and 2014, with over 30,000 reported cases [1]. From French Polynesia, the disease spread to New Caledonia, The Cook Islands and Eastern Islands [22]. This recent and still-ravaging outbreak of zika began in April 2015, in Brazil [10], and has spread to many other countries in south and central Americas and the Caribbean, with over 140,000 suspected and confirmed cases by the end of February 2016. In the United states, Zika has been detected in Florida, New York city, Texas, and other places ([4], [18], [8]).

At present, there is no known cure for zika virus disease. Only treatments are administered based on the symptoms for which the disease is known. Also, the idea using bed nets to reduce mosquitoes bites cannot be applied in this case because aedes aegypti mosquitoes are day-biters. Also, the use of physical and chemical control methods is not advisable since they have not been effective in controlling other mosquito-borne diseases such as malaria and dengue. Hence, it seems that the only feasible option is to apply biological control method to reduce the spread of the disease.

Reasonable number of models have been proposed recently on the study of zika virus disease. There are models that focus on understanding the transmission dynamics of the disease, as can be seen in [20], [24], [5], [15] and others. There are also models which incorporate sexual transmission in the dynamics of the disease, as in [25], [9] and [26]. A model that is based on biological control of the disease can be found in [28], where the use of wolbachia-infected aedes aegypti mosquitoes was adopted as bio-control for the disease. This method has been effectively applied in controlling similar mosquito borne flavivirus diseases such as dengue and West Nile, for example see [23]. Our model in this work is also based on this innovative mosquito control method.

The rest of this work is organized as follows: In section 2, we present the idea behind using wolbachia-infected mosquitoes as agent for controlling the spread of zika virus disease. This is followed by section 3 where we presented our model with its basic assumptions. In section 4, we estimated the control reproduction number of the disease and showed that it is below unity. Finally, in section 5, we solved the model and obtained simulation results. The results were also discussed in this section.

2 Wolbachia, Aedes Aegypti and Zika Virus Disease

Wolbachia is a group of bacteria naturally found in reproductive tissues of some arthropods like some mosquitoes. They are transmitted maternally through the cytoplasm of eggs of their hosts. Some strains of *wolbachia* such as *w*Mel reduce the ability of the host insects to transmit disease-causing pathogens to humans. This is the idea behind using wolbachia-infected mosquitoes as bio-control for zika virus disease. Aedes aegypti is not known to be a natural carrier of wolbachia. The mosquitoes have to be manually infected with the bacteria in the laboratory and released in the zika-endemic area in order to fight zika virus disease [13]. Wolbachia helps to fight zika virus disease by increasing the incubation period(or reduces the incubation rate) of the virus in the infected mosquitoes. Since the adult life of the mosquito is short (about 14 days), most of the mosquitoes carrying the virus die before they become infectious. Hence, the infected *wolbachia*-carrier mosquitoes may not transmit the virus to humans through their bites. Also *wolbachia* induces cytoplasmic incompatibility(CI), which helps its host mosquito to eliminate the wolbachia-free aedes aegypti mosquitoes in the wild [14]. CI is a phenomenon that prevents the formation of embryos of egg (non hatching of eggs) when *wolbachia*-carrier male mosquitoes mate with *wolbachia*-free female mosquitoes or mate with female mosquitoes that carry a different *wolbachia* strain [17]. The effect of CI on the mosquitoes' reproduction process is summarized below.

- wolbachia-free males mate with wolbachia-free females to produce wolbachia-free offspring.
- wolbachia-free males mate with wolbachia-carrier females to produce wolbachia-carrier offspring.
- wolbachia-carrier male mate with wolbachia-free females to produce non-viable eggs.
- wolbachia-carrier male mate with wolbachia-carrier females to produce wolbachia-carrier offsrpring.

Hence, with cytoplasmic incompatibility, more *wolbachia*-carrier mosquitoes are produced [11], and these wolbachia-carrier mosquitoes will have very low probability to transmit the virus to humans, therefore limiting the spread of the disease.

3 THE MATHEMATICAL MODEL

Humans may contract zika virus when bitten by infectious female Aedes *aegypti* mosquito or when infected human passes the virus to uninfected human through unsafe sex, unsafe blood transfusion or perinatal transmission from mother to child. On the other hand, transmission of zika virus from human to mosquito occurs when an adult, uninfected female Aedes *aegypti* mosquito bites human to suck blood. If the human is already infected with the virus, he may pass it to the mosquito. The mosquito once infected, remains so and continues to infect humans throughout its life time. The model is made up of three major populations;

human population, adult female wolbachia-free aedes *aegypti* population and adult female wolbachia-carrier aedes *aegypti* population used as control.

3.1 Zika Dynamics in Human Population

The total human population, $N_H(t)$, at any time, t, is divided into 8 compartments or classes, namely, (i) the susceptible class, $S_H(t)$, (ii) the latent or the exposed class, $E_H(t)$, (iii) the symptomatically infectious class, $I_{Hs}(t)$, (iv) the asymptomatically infectious class, $I_{Has}(t)$, (v) the treatment class, $I_T(t)$, (vi) the non- treatment class, $I_{NT}(t)$, (vii)the partially recovered class, $R_{H1}(t)$, (viii) the totally recovered class, $R_{H2}(t)$. The following assumptions are made on the transmission dynamics of zika virus disease in the human population:

- Individuals in the human population are recruited into the susceptible class either through migration into the zika endemic area at the rate, Π_H, or through birth of zika virus-free offspring at the rate μ_H.
- The susceptible class acquires zika virus either through infectious wolbachia-free mosquito bites, with probability, α_{MH}, or through humans in the infectious classes; treatment class, non-treatment class and partially recovered class, with probability, α_{HH}, to move to the exposed class.
- The susceptible humans may also contract the virus through the bites of wolbachia-carrier mosquitoes with a very negligible probability of infection, $\alpha_{MwH} \ll \alpha_{MH}$.
- The exposed class becomes infectious at the incubation rate β_H, to become either asymptomatically or symptomatically infectious, in the proportions v and $(1-v)$, respectively.
- Due to transmission of zika virus from infected pregnant mothers to their offspring, we assume that the proportion δ, of the young ones does not carry the virus and, hence is transferred to the susceptible class, $S_H(t)$, while the infected proportion $(1-\delta)$, moves to the exposed class, $E_H(t)$.
- The proportion, ω of the symptomatically infectious class receives treatment at the rate τ, to be in the treatment class, $I_T(t)$, whereas $(1-\omega)$ receives no treatment, and stays in the non-treatment class, $I_{NT}(t)$.
- The treatment and the non-treatment classes recover partially at the rates, ν_1, and ν_2, respectively, to move to the partially recovered class, $R_{H1}(t)$.
- After some period of time, the partially recovered and the asymptomatically infectious classes recover fully at the rates γ_1 and γ_2 respectively, to be in the totally recovered class, $R_{H2}(t)$. An individual remains in the totally recovered class, and cannot be reinfected with the virus by any means until natural death occurs.

- All the classes in the human population benefit from the natural mortality rate σ_H, whereas only the diseased classes are affected by zika-induced death at the rate σ_H^I, which is negligible.

3.2 Zika Dynamics in the Aedes aegypti Population

The population of wolbachia-free adult female aedes *aegypti* mosquitoes is grouped into 3 classes namely; (i)the susceptible mosquitoes, $S_M(t)$, (ii) the exposed mosquitoes, $E_M(t)$ and (iii)the infectious mosquitoes, $I_M(t)$.

- The wolbachia-free male mosquitoes mate with their female counterparts in the wolbachia-free and wolbachia-carrier mosquito populations to produce wolbachia-free and wolbachia-carrier offspring, respectively.
- The female wolbachia-free mosquitoes join the susceptible class through migration at the rate Π_M, or through oviposition at the rate μ_M.
- To model the effect of cytoplasmic incompatibility, we assume that the proportion q, of the eggs produced by the female wolbachia-free mosquitoes are viable, while $(1-q)$ are non-viable.
- Susceptible mosquitoes contract zika virus when they bite humans in the infectious classes at the biting rate b_1, with probability of infection α_{HM}, and move to the exposed class.
- After some period of time (incubation period) in the exposed class, the mosquitoes become infectious and move to the infectious class at the rate, β_M.
- The mosquitoes remain infectious throughout their lifetime until they die naturally at the rate, σ_M.

Similarly, the female adult wolbachia-carrier aedes *aegypti* mosquitoes are grouped in the same manner, with the following compartments, the susceptible wolbachia-carrier class; $S_{Mw}(t)$, the exposed wolbachia-carrier class; $E_{Mw}(t)$, and the infectious wolbachia-carrier class; $I_{Mw}(t)$,
The dynamics of zika virus disease in the wolbachia-carrier mosquito population is similar to that of the wolbachia-free mosquitoes, except at the infectious stage where the probability of the wolbachia-carrier mosquitoes to transmit the virus to the susceptible humans is negligible.

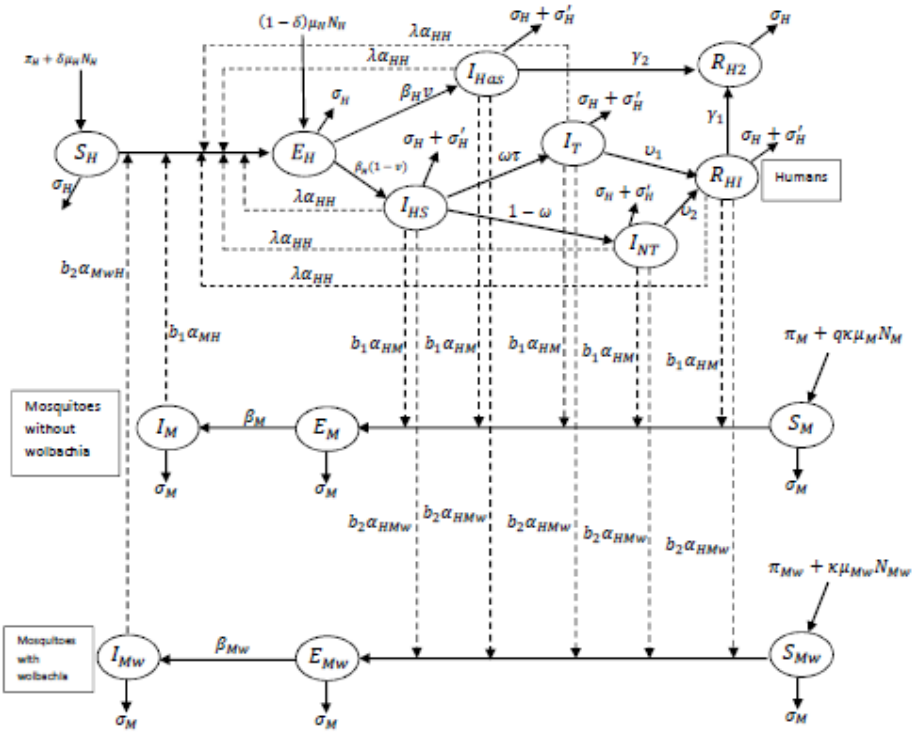

Fig 1: Flow Diagram for the Disease Transmission and Control

The assumptions above and the flow diagram(figure 1) lead to the following system of ordinary differential equations as our model for the transmission and control of zika virus disease.

$$\begin{aligned}
\frac{dS_H(t)}{dt} &= \Pi_H + \delta\mu_H N_H - \left(b_1\alpha_{MH}\frac{I_M}{N_M} + b_2\alpha_{MwH}\frac{I_{Mw}}{N_{Mw}} + \lambda\alpha_{HH}F(N_H) + \sigma_H\right)S_H,\\
\frac{dE_H(t)}{dt} &= (1-\delta)\mu_H N_H + \left(b_1\alpha_{MH}\frac{I_M}{N_M} + b_2\alpha_{MwH}\frac{I_{Mw}}{N_{Mw}} + \lambda\alpha_{HH}F(N_H)\right)S_H - (\beta_H + \sigma_H)E_H\\
\frac{dI_{Has}(t)}{dt} &= v\beta_H E_H - (\gamma_2 + \sigma_H + \sigma'_H)I_{Has},\\
\frac{dI_{Hs}(t)}{dt} &= (1-v)\beta_H E_H - \left(\tau\omega + 1 - \omega + \sigma_H + \sigma'_H\right)I_{Hs},\\
\frac{dI_T(t)}{dt} &= \tau\omega I_{Hs} - (\nu_1 + \sigma_H + \sigma'_H)I_T,\\
\frac{dI_{NT}(t)}{dt} &= (1-\omega)I_{Hs} - (\nu_2 + \sigma_H + \sigma'_H)I_{NT},\\
\frac{dR_{H1}(t)}{dt} &= \nu_1 I_T + \nu_2 I_{NT} - (\gamma_1 + \sigma_H + \sigma'_H)R_{H1},\\
\frac{dR_{H2}(t)}{dt} &= \gamma_1 R_{H1} + \gamma_2 I_{Has} - \sigma_H R_{H2},\\
\frac{dS_M(t)}{dt} &= \Pi_M + q\kappa\mu_M N_M - b_1\alpha_{HM}S_M(t)F(N_H) - \sigma_M S_M(t),\\
\frac{dE_M(t)}{dt} &= b_1\alpha_{HM}S_M(t)F(N_H) - (\beta_M + \sigma_M)E_M(t),\\
\frac{dI_M(t)}{dt} &= \beta_M E_M(t) - \sigma_M I_M(t),\\
\frac{dS_{Mw}(t)}{dt} &= \Pi_{Mw} + \kappa\mu_{Mw}N_{Mw} - b_2\alpha_{HMw}S_{Mw}(t)F(N_H) - \sigma_M S_{Mw}(t),\\
\frac{dE_{Mw}(t)}{dt} &= b_2\alpha_{HMw}S_{Mw}(t)F(N_H) - (\beta_{Mw} + \sigma_M)E_{Mw}(t),\\
\frac{dI_{Mw}(t)}{dt} &= \beta_{Mw}E_{Mw}(t) - \sigma_M I_{Mw}(t),
\end{aligned} \tag{1}$$

where

$$F(N_H) = \left(\frac{I_{Has} + I_{Hs} + I_T + I_{NT} + R_{H1}}{N_H}\right)$$

The initial conditions are $S_H(0) = S_H^0, E_H(0) = E_H^0, I_{Has}(0) = I_{Has}^0, I_{Hs}(0) = I_{Hs}^0, I_T(0) = I_T^0, I_{NT}(0) = I_{NT}^0, R_{H1}(0) = R_{H1}^0, R_{H2}(0) = R_{H2}^0, S_M(0) = S_M^0, E_M(0) = E_M^0, I_M(0) = I_M^0, S_{Mw}(0) = S_{Mw}^0, E_{Mw}(0) = E_{Mw}^0, I_{Mw}(0) = I_{Mw}^0$, which we assume to be all nonnegative quantities. From (1), we see that the total populations of human, wolbachia-free and wolbachia-carrier mosquitoes, respectively, satisfy the equations

$$\begin{aligned}
\frac{dN_H}{dt} &= \Pi_H + (\mu_H - \sigma_H)N_H - \sigma'_H N'_H\\
\frac{dN_M}{dt} &= \Pi_M + (q\kappa\mu_M - \sigma_M)N_M\\
\frac{dN_{Mw}}{dt} &= \Pi_{Mw} + (\kappa\mu_{Mw} - \sigma_M)N_{Mw}
\end{aligned} \tag{2}$$

The domain of existence of the solution to the system can be described as

$$\mathcal{D} = \mathcal{D}_1 \cup \mathcal{D}_2 \cup \mathcal{D}_3$$

where

$$\begin{aligned}\mathcal{D}_1 &= \{(S_H, E_M, I_{Has}, I_{Hs}, I_{NT}, I_T, R_{H1}, R_{H2}) \in \mathbb{R}_+^8 \\ &\quad \big| S_H + E_M + I_{as} + I_s + I_{NT} + I_T + R_{H1} + R_{H2} \leq N_H\} \\ \mathcal{D}_2 &= \{(S_M, E_M, I_M) \in \mathbb{R}_+^3 \big| S_M + E_M + I_M \leq N_M\} \\ \mathcal{D}_3 &= \{(S_{Mw}, E_{Mw}, I_{Mw}) \in \mathbb{R}_+^3 \big| S_{Mw} + E_{Mw} + I_{Mw} \leq N_{Mw}\}\end{aligned} \tag{3}$$

Using $x_1 = \frac{S_H}{N_H}, x_2 = \frac{E_H}{N_H}, x_3 = \frac{I_{Has}}{N_H}, \cdots, x_8 = \frac{R_{H2}}{N_H}, y_1 = \frac{S_M}{N_M}, y_2 = \frac{E_M}{N_M}, y_3 = \frac{I_M}{N_M}, z_1 = \frac{S_{Mw}}{N_{Mw}}, z_2 = \frac{E_M}{N_{Mw}}, z_3 = \frac{I_M}{N_{Mw}}$ and the product rule, we get the dimensionless form of (1):

$$\begin{aligned}\frac{dx_1}{dt} &= \frac{\Pi_H}{N_H} + \delta\mu_H - f(y_3, z_3, x_d)x_1 - \left(\frac{\Pi_H}{N_H} + \mu_H - \sigma_H' x_d\right) x_1 \\ \frac{dx_2}{dt} &= (1-\delta)\mu_H + f(y_3, z_3, x_d)x_1 - \left(\beta_H + \frac{\Pi_H}{N_H} + \mu_H - \sigma_H' x_d\right) x_2 \\ \frac{dx_3}{dt} &= v\beta_H x_2 - \left(\gamma_2 + \frac{\Pi_H}{N_H} + \sigma_H' + \mu_H - \sigma_H' x_d\right) x_3 \\ \frac{dx_4}{dt} &= (1-v)\beta_H x_2 - \left(\tau\omega + 1 - \omega + \frac{\Pi_H}{N_H} + \sigma_H' + \mu_H - \sigma_H' x_d\right) x_4 \\ \frac{dx_5}{dt} &= \tau\omega x_4 - \left(\nu_1 + \frac{\Pi_H}{N_H} + \sigma_H' + \mu_H - \sigma_H' x_d\right) x_5 \\ \frac{dx_6}{dt} &= (1-\omega)x_4 - \left(\nu_2 + \frac{\Pi_H}{N_H} + \sigma_H' + \mu_H - \sigma_H' x_d\right) x_6 \\ \frac{dx_7}{dt} &= \nu_1 x_5 + \nu_2 x_6 - \left(\gamma_1 + \frac{\Pi_H}{N_H} + \sigma_H' + \mu_H - \sigma_H' x_d\right) x_7 \\ \frac{dx_8}{dt} &= \gamma_1 x_7 + \gamma_2 x_3 - \left(\frac{\Pi_H}{N_H} + \mu_H - \sigma_H' x_d\right) x_8 \\ \frac{dy_1}{dt} &= \frac{\Pi_M}{N_M} + q\kappa\mu_M - \left(b_1\alpha_{HM} x_d + q\kappa\mu_M + \frac{\Pi_M}{N_M}\right) y_1 \\ \frac{dy_2}{dt} &= b_1\alpha_{HM} x_d y_1 - \left(\beta_M + q\kappa\mu_M + \frac{\Pi_M}{N_M}\right) y_2 \\ \frac{dy_3}{dt} &= \beta_M y_2 - \left(q\kappa\mu_M + \frac{\Pi_M}{N_M}\right) y_3 \\ \frac{dz_1}{dt} &= \frac{\Pi_{Mw}}{N_{Mw}} + \kappa\mu_{Mw} - \left(b_2\alpha_{HMw} x_d + \kappa\mu_{Mw} + \frac{\Pi_{Mw}}{N_{Mw}}\right) z_1 \\ \frac{dz_2}{dt} &= b_2\alpha_{HMw} x_d z_1 - \left(\beta_{Mw} + \kappa\mu_{Mw} + \frac{\Pi_{Mw}}{N_{Mw}}\right) z_2 \\ \frac{dz_3}{dt} &= \beta_{Mw} z_2 - \left(\kappa\mu_{Mw} + \frac{\Pi_{Mw}}{N_{Mw}}\right) z_3\end{aligned} \tag{4}$$

where $x_d = x_3 + x_4 + x_5 + x_6 + x_7$ and $f(y_3, z_3, x_d) = b_1\alpha_{MH} y_3 + b_2\alpha_{MwH} z_3 + \lambda\alpha_{HH} x_d$

The initial conditions are $x_1(0) = x_{10}, x_2(0) = x_{20}, x_3(0) = x_{30}, x_4(0) = x_{40}, x_5(0) = x_{50}, x_6(0) = x_{60}$, $x_7(0) = x_{70}, x_8(0) = x_{80}, y_1(0) = y_{10}, \quad y_2(0) = y_{20}, y_3(0) = y_{30}, z_1(0) = z_{10}, z_2(0) = z_{20}, z_3(0) = z_{30}$, which we assume to be all non-negative quantities.

The system (4) can also be written as

$$X'(t) = f(X(t)) \tag{5}$$

where $X = (x_1, x_2, x_3, x_4, x_5, x_6, x_7, x_8, y_1, y_2, y_3, z_1, z_2, z_3)^T$, and

$$f(X(t)) = \begin{pmatrix} \frac{\Pi_H}{N_H} + \delta\mu_H - f(y_3, z_3, x_d)x_1 - \left(\frac{\Pi_H}{N_H} + \mu_H - \sigma'_H x_d\right) x_1 \\ (1-\delta)\mu_H + f(y_3, z_3, x_d)x_1 - \left(\beta_H + \frac{\Pi_H}{N_H} + \mu_H - \sigma'_H x_d\right) x_2 \\ v\beta_H x_2 - \left(\gamma_2 + \frac{\Pi_H}{N_H} + \sigma'_H + \mu_H - \sigma'_H x_d\right) x_3 \\ (1-v)\beta_H x_2 - \left(\tau\omega + 1 - \omega + \frac{\Pi_H}{N_H} + \sigma'_H + \mu_H - \sigma'_H x_d\right) x_4 \\ \tau\omega x_4 - \left(\nu_1 + \frac{\Pi_H}{N_H} + \sigma'_H + \mu_H - \sigma'_H x_d\right) x_5 \\ (1-\omega)x_4 - \left(\nu_2 + \frac{\Pi_H}{N_H} + \sigma'_H + \mu_H - \sigma'_H x_d\right) x_6 \\ \nu_1 x_5 + \nu_2 x_6 - \left(\gamma_1 + \frac{\Pi_H}{N_H} + \sigma'_H + \mu_H - \sigma'_H x_d\right) x_7 \\ \gamma_1 x_7 + \gamma_2 x_3 - \left(\frac{\Pi_H}{N_H} + \mu_H - \sigma'_H x_d\right) x_8 \\ \frac{\Pi_M}{N_M} + q\kappa\mu_M - \left(b_1\alpha_{HM}x_d + q\kappa\mu_M + \frac{\Pi_M}{N_M}\right) y_1 \\ b_1\alpha_{HM}x_d y_1 - \left(\beta_M + q\kappa\mu_M + \frac{\Pi_M}{N_M}\right) y_2 \\ \beta_M y_2 - \left(q\kappa\mu_M + \frac{\Pi_M}{N_M}\right) y_3 \\ \frac{\Pi_{Mw}}{N_{Mw}} + \kappa\mu_{Mw} - \left(b_2\alpha_{HMw}x_d + \kappa\mu_{Mw} + \frac{\Pi_{Mw}}{N_{Mw}}\right) z_1 \\ b_2\alpha_{HMw}x_d z_1 - \left(\beta_{Mw} + \kappa\mu_{Mw} + \frac{\Pi_{Mw}}{N_{Mw}}\right) z_2 \\ \beta_{Mw} z_2 - \left(\kappa\mu_{Mw} + \frac{\Pi_{Mw}}{N_{Mw}}\right) z_3 \end{pmatrix} \tag{6}$$

4 Disease-Free Equilibrium and The Control Reproduction Number

From (5), the disease-free equilibrium of (4) is the point E_0 such that $f(E_0) = 0$, when there is no disease infection in the population. That is the solution to $f(X(t)) = 0$ given that $x_2 = x_3 = x_4 = x_5 = x_6 = x_7 = y_2 = y_3 = z_2 = z_3 = 0$.

Using this definition, we have that $E_0 = (1, 0, 0, 0, 0, 0, 0, 0, 1, 0, 0, 1, 0, 0)$. Also, at disease-free equilibrium, the total human, wolbachia-free mosquitoes and wolbachia-carrier mosquitoes populations can be obtained as

$$N_H = \frac{\Pi_H}{\sigma_H - \mu_H}, \; N_M = \frac{\Pi_M}{\sigma_M - q\kappa\mu_M}, \; N_{Mw} = \frac{\Pi_{Mw}}{\sigma_M - \kappa\mu_{Mw}}$$

respectively.

The basic reproduction number is the average number of individuals that will be infected by a typical infectious individual throughout his infectious lifetime when introduced in a purely susceptible population [30]. Generally denoted by $\mathcal{R}_0$, the basic reproduction number is considered as threshold parameter which determines the potential for disease outbreak to continue or to fizzle out overtime. This is because if $\mathcal{R}_0 < 1$, then on average, an infected individual produces less than one new infected person throughout the course of its infectious period, and the disease infection cannot spread in the population. Conversely, if $\mathcal{R}_0 > 1$, then each infected individual produces on average, more than one new infectious persons and the disease grows and invade the entire susceptible population.

We shall determine the control reproduction number $\mathcal{R}_c$ which is the basic reproduction number in the

presence of the control measure. To determine $\mathcal{R}_c$ we shall adopt the next generation matrix approach introduced in [6],where reproduction number was estimated as the dominant eigenvalue or the spectral radius of the next generation matrix.

If we write $f(X(t))$ in the form

$$f(X(t)) = \mathcal{F}_i(x) - \mathcal{V}_i(x) \tag{7}$$

where $\mathcal{F}_i(x)$ are the terms which have the rate of appearance of new infections in compartment i, while $\mathcal{V}_i(x)$ are the terms which have the rate of transfer of individuals into and out of compartment i by any other means including death, then the next generation matrix is given by $\mathcal{K} = FV^{-1}$, where $F = \frac{\partial \mathcal{F}(x)}{\partial x}|_{E_0}$ and $V = \frac{\partial \mathcal{V}(x)}{\partial x}|_{E_0}$ and the control reproduction number is therefore the spectral radius of FV^{-1}; $\mathcal{R}_c = \rho(FV^{-1})$.

Theorem 1 *The next generation matrix of the model (1) or equivalently (4) has the form*

$$\mathcal{K} = \begin{pmatrix} \mathcal{K}_{HH} & \mathcal{K}_{MH} & \mathcal{K}_{MwH} \\ \mathcal{K}_{HM} & 0 & 0 \\ \mathcal{K}_{HMw} & 0 & 0 \end{pmatrix} \tag{8}$$

and the control reproduction number is

$$\mathcal{R}_c = \frac{\mathcal{K}_{HH}}{2} + \frac{\sqrt{\mathcal{K}_{HH}^2 + 4(\mathcal{K}_{MH}\mathcal{K}_{HM} + \mathcal{K}_{MwH}\mathcal{K}_{HMw})}}{2} \tag{9}$$

where

$\mathcal{K}_{HH}$ *= the number of humans that one infectious human can infect throughout his infectious lifetime, when introduced in a purely susceptible human population.*

$\mathcal{K}_{HM}$ *= the number of wolbachia-free mosquitoes that one infectious human can infect throughout his infectious lifetime, when introduced in a purely susceptible wolbachia-free mosquito population.*

$\mathcal{K}_{HMw}$ *= the number of wolbachia-carrier mosquitoes that one infectious human can infect throughout his infectious lifetime, when introduced in a purely susceptible wolbachia-carrier mosquito population.*

$\mathcal{K}_{MH}$ *= the number of humans that one infectious wolbachia-free mosquito can infect throughout its infectious lifetime, when introduced in a purely susceptible human population.*

$\mathcal{K}_{MwH}$ *= the number of humans that one infectious wolbachia-carrier mosquito can infect throughout its infectious lifetime, when introduced in a purely susceptible human population.*

Proof: The form of the next generation matrix (8) is informed by the following major assumptions made on the transmission dynamics and control of the disease:

- three populations are involved in the dynamics of the disease,
- humans can transmit the virus to other humans in the human population through sexual relationship and perinatal transmission,

- humans can infect mosquitoes in the wolbachia-free and wolbachia-carrier mosquitoes populations,
- wolbachia-free and wolbachia-carrier mosquitoes can bite and infect humans, and
- there is no vertical transmission of zika virus from females mosquitoes to their offspring,

To derive (8), we first put $f(X(t))$ in the form (7), where

$$\mathcal{F}_i(x) = \begin{bmatrix} (1-\delta)\mu_H + f(y_3, z_3, x_d)x_1 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ b_1\alpha_{HM}x_d y_1 \\ 0 \\ b_2\alpha_{HMw}x_d z_1 \\ 0 \end{bmatrix}, \mathcal{V}_i(x) = \begin{bmatrix} \left(\beta_H + \frac{\Pi_H}{N_H} + \mu_H - \sigma'_H x_d\right) x_2 \\ -v\beta_H x_2 + \left(\gamma_2 + \frac{\Pi_H}{N_H} + \sigma'_H + \mu_H - \sigma'_H x_d\right) x_3 \\ -(1-v)\beta_H x_2 + \left(\tau\omega + 1 - \omega + \frac{\Pi_H}{N_H} + \sigma'_H + \mu_H - \sigma'_H x_d\right) x_4 \\ -\tau\omega x_4 + \left(\nu_1 + \frac{\Pi_H}{N_H} + \sigma'_H + \mu_H - \sigma'_H x_d\right) x_5 \\ -(1-\omega)x_4 + \left(\nu_2 + \frac{\Pi_H}{N_H} + \sigma'_H + \mu_H - \sigma'_H x_d\right) x_6 \\ -\nu_1 x_5 - \nu_2 x_6 + \left(\gamma_1 + \frac{\Pi_H}{N_H} + \sigma'_H + \mu_H - \sigma'_H x_d\right) x_7 \\ \left(\beta_M + q\kappa\mu_M + \frac{\Pi_M}{N_M}\right) y_2 \\ -\beta_M y_2 + \left(q\kappa\mu_M + \frac{\Pi_M}{N_M}\right) y_3 \\ \left(\beta_{Mw} + \kappa\mu_{Mw} + \frac{\Pi_{Mw}}{N_{Mw}}\right) z_2 \\ -\beta_{Mw} z_2 + \left(\kappa\mu_{Mw} + \frac{\Pi_{Mw}}{N_{Mw}}\right) z_3 \end{bmatrix}$$

Therefore,

$$F = \begin{pmatrix} 0 & F_1 & F_2 & F_3 & F_4 & F_5 & 0 & F_6 & 0 & F_7 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & F_8 & F_9 & F_{10} & F_{11} & F_{12} & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & F_{13} & F_{14} & F_{15} & F_{16} & F_{17} & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \end{pmatrix} \tag{10}$$

where $F_i = \lambda\alpha_{HH}, i = 1,2,3,4,5.\ F_6 = b_1\alpha_{MH},\ F_7 = b_2\alpha_{MwH},\ F_j = b_1\alpha_{HM}, j = 8,9,10,11,12.$

$F_k = b_2\alpha_{HMw}, k = 13,14,15,16,17$

$$V = \begin{pmatrix} V_1 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ -V_2 & V_3 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ -V_4 & 0 & V_5 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & -V_6 & V_7 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & -V_8 & 0 & V_9 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & -V_{10} & -V_{11} & V_{12} & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & V_{13} & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & -V_{14} & V_{15} & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & V_{16} & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & -V_{17} & V_{18} \end{pmatrix} \tag{11}$$

where

$V_1 = \beta_H + \sigma_H,\ \ V_2 = v\beta_H,\ \ V_3 = \gamma_2 + \sigma_H + \sigma'_H, V_4 = (1-v)\beta_H,\ \ V_5 = \tau\omega + 1 - \omega + \sigma_H + \sigma'_H,$

$V_6 = \tau\omega, V_7 = \nu_1 + \sigma_H + \sigma'_H,\ V_8 = (1-\omega),\ V_9 = \nu_2 + \sigma_H + \sigma'_H, V_{10} = \nu_1, V_{11} = \nu_2,$

$V_{12} = (\gamma_1 + \sigma_H + \sigma'_H), V_{13} = \beta_M + \sigma_M, V_{14} = \beta_M, V_{15} = \sigma_M, V_{16} = \beta_{Mw} + \sigma_M, V_{17} = \beta_{Mw},$

$V_{18} = \sigma_M.$

The inverse of V is non-negative and is given by

$$V^{-1} = \begin{pmatrix} V_1^\bullet & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ V_2^\bullet & V_3^\bullet & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ V_4^\bullet & 0 & V_5^\bullet & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ V_6^\bullet & 0 & V_7^\bullet & V_8^\bullet & 0 & 0 & 0 & 0 & 0 & 0 \\ V_9^\bullet & 0 & V_{10}^\bullet & 0 & V_{11}^\bullet & 0 & 0 & 0 & 0 & 0 \\ V_{12}^\bullet & 0 & V_{13}^\bullet & V_{14}^\bullet & V_{15}^\bullet & V_{16}^\bullet & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & V_{17}^\bullet & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & V_{18}^\bullet & V_{19}^\bullet & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & V_{20}^\bullet & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & V_{21}^\bullet & V_{22}^\bullet \end{pmatrix} \tag{12}$$

where

$$V_1^\bullet = \frac{1}{(\beta_H + \sigma_H)},\ \ V_2^\bullet = \frac{v\beta_H}{(\beta_H + \sigma_H)(\gamma_2 + \sigma_H + \sigma'_H)}, V_3^\bullet = \frac{1}{(\gamma_2 + \sigma_H + \sigma'_H)}$$

$$, V_4^\bullet = \frac{(1-v)\beta_H}{(\beta_H + \sigma_H)(\tau\omega + 1 - \omega + \sigma_H + \sigma'_H)}, V_5^\bullet = \frac{1}{(\tau\omega + 1 - \omega + \sigma_H + \sigma'_H)},$$

$$V_6^\bullet = \frac{(1-v)\beta_H\tau\omega}{(\beta_H + \sigma_H)(\tau\omega + 1 - \omega + \sigma_H + \sigma'_H)(\nu_1 + \sigma_H + \sigma'_H)}, V_7^\bullet = \frac{\tau\omega}{(\tau\omega + 1 - \omega + \sigma_H + \sigma'_H)(\nu_1 + \sigma_H + \sigma'_H)}$$

$$V_8^\bullet = \frac{1}{(\nu_1+\sigma_H+\sigma_H')}, V_9^\bullet = \frac{\beta_H(1-v)(1-\omega)}{(\beta_H+\sigma_H)(\tau\omega+1-\omega+\sigma_H+\sigma_H')(\nu_2+\sigma_H+\sigma_H')}$$

$$V_{10}^\bullet = \frac{(1-\omega)}{(\tau\omega+1-\omega+\sigma_H+\sigma_H')(\nu_2+\sigma_H+\sigma_H')}, V_{11}^\bullet = \frac{1}{(\nu_2+\sigma_H+\sigma_H')}$$

$$V_{12}^\bullet = \frac{\tau\omega\nu_1(1-v)\beta_H(\nu_2+\sigma_H+\sigma_H')+\nu_2(1-v)\beta_H(1-\omega)(\nu_1+\sigma_H+\sigma_H')}{(\beta_H+\sigma_H)(\gamma_1+\sigma_H+\sigma_H')(\tau\omega+1-\omega+\sigma_H+\sigma_H')(\nu_2+\sigma_H+\sigma_H')(\nu_1+\sigma_H+\sigma_H')}$$

$$V_{13}^\bullet = \frac{(\nu_1+\nu_2)\tau\omega}{(\gamma_1+\sigma_H+\sigma_H')(\tau\omega+1-\omega+\sigma_H+\sigma_H')(\nu_1+\sigma_H+\sigma_H')}, V_{14}^\bullet = \frac{\nu_1}{(\gamma_1+\sigma_H+\sigma_H')(\nu_1+\sigma_H+\sigma_H')}$$

$$V_{15}^\bullet = \frac{\nu_2}{(\gamma_1+\sigma_H+\sigma_H')(\nu_1+\sigma_H+\sigma_H')}, \quad V_{16}^\bullet = \frac{1}{(\gamma_1+\sigma_H+\sigma_H')}, V_{17}^\bullet = \frac{1}{(\beta_M+\sigma_M)}$$

$$V_{18}^\bullet = \frac{\beta_M}{(\beta_M+\sigma_M)\sigma_M}, V_{19}^\bullet = \frac{1}{\sigma_M}, \quad V_{20}^\bullet = \frac{1}{(\beta_{Mw}+\sigma_M)}, \quad V_{21}^\bullet = \frac{\beta_{Mw}}{(\beta_{Mw}+\sigma_M)\sigma_M}, \quad V_{22}^\bullet = \frac{1}{\sigma_M}$$

Then, the next generation matrix is

$$\mathcal{K} = \begin{pmatrix} K_{11} & K_{12} & K_{13} & K_{14} & K_{15} & K_{16} & K_{17} & K_{18} & K_{19} & K_{110} \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ K_{71} & K_{72} & K_{73} & K_{74} & K_{75} & K_{76} & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ K_{91} & K_{92} & K_{93} & K_{94} & K_{95} & K_{96} & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \end{pmatrix} \tag{13}$$

To get (8), we pre- and post-multiply (13) with an auxiliary matrix of the form

$$A = \begin{pmatrix} 1 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 1 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 1 & 0 \end{pmatrix}^t$$

The auxiliary matrix A will help to single out the rows and columns of (13) that are relevant in the determination of the control reproduction number $\mathcal{R}_c$. The dimension of A is $m \times n$, with $n < m$, such that m is the number of disease states and n is the number of states at infection, with 1 in the corresponding row where there is state at infection, and zero elsewhere. Then, the reduced next generation matrix becomes $\mathcal{K}_s = \begin{pmatrix} K_{11}' & K_{17}' & K_{19}' \\ K_{71}' & 0 & 0 \\ K_{91}' & 0 & 0 \end{pmatrix}$ which is equivalent to (8), where

$$K_{11}' \equiv K_{HH} = \mathcal{A}_1 \left[\frac{v}{(\gamma_2+\sigma_H+\sigma_H')} + \frac{1-v}{(\tau\omega+1-\omega+\sigma_H+\sigma_H')} \right]$$

$$+\mathcal{A}_1\frac{\nu_1\tau\omega(1-v)\left(\nu_2+\sigma_H+\sigma'_H\right)+\nu_2(1-v)(1-\omega)\left(\nu_1+\sigma_H+\sigma'_H\right)}{\left(\gamma_1+\sigma_H+\sigma'_H\right)\left(\tau\omega+1-\omega+\sigma_H+\sigma'_H\right)\left(\nu_1+\sigma_H+\sigma'_H\right)\left(\nu_2+\sigma_H+\sigma'_H\right)}$$

$$+\mathcal{A}_1\left[\frac{(1-v)\tau\omega}{\left(\tau\omega+1-\omega+\sigma_H+\sigma'_H\right)\left(\nu_1+\sigma_H+\sigma'_H\right)}+\frac{(1-v)(1-\omega)}{\left(\tau\omega+1-\omega+\sigma_H+\sigma'_H\right)\left(\nu_2+\sigma_H+\sigma'_H\right)}\right]$$

$$K'_{71}\equiv K_{HM}=\mathcal{A}_2\frac{\nu_1\tau\omega(1-v)\left(\nu_2+\sigma_H+\sigma'_H\right)+\nu_2(1-v)(1-\omega)\left(\nu_1+\sigma_H+\sigma'_H\right)}{\left(\gamma_1+\sigma_H+\sigma'_H\right)\left(\tau\omega+1-\omega+\sigma_H+\sigma'_H\right)\left(\nu_1+\sigma_H+\sigma'_H\right)\left(\nu_2+\sigma_H+\sigma'_H\right)}$$

$$+\mathcal{A}_2\left[\frac{(1-v)}{\left(\tau\omega+1-\omega+\sigma_H+\sigma'_H\right)}+\frac{v}{\left(\gamma_2+\sigma_H+\sigma'_H\right)}\right]$$

$$+\mathcal{A}_2\left[\frac{(1-v)\tau\omega}{\left(\tau\omega+1-\omega+\sigma_H+\sigma'_H\right)\left(\nu_1+\sigma_H+\sigma'_H\right)}+\frac{(1-v)(1-\omega)}{\left(\tau\omega+1-\omega+\sigma_H+\sigma'_H\right)\left(\nu_2+\sigma_H+\sigma'_H\right)}\right]$$

$$K'_{91}\equiv K_{HMw}=\mathcal{A}_3\frac{\nu_1\tau\omega(1-v)\left(\nu_2+\sigma_H+\sigma'_H\right)+\nu_2(1-v)(1-\omega)\left(\nu_1+\sigma_H+\sigma'_H\right)}{\left(\gamma_1+\sigma_H+\sigma'_H\right)\left(\tau\omega+1-\omega+\sigma_H+\sigma'_H\right)\left(\nu_1+\sigma_H+\sigma'_H\right)\left(\nu_2+\sigma_H+\sigma'_H\right)}$$

$$+\mathcal{A}_3\left[\frac{(1-v)}{\left(\tau\omega+1-\omega+\sigma_H+\sigma'_H\right)}+\frac{v}{\left(\gamma_2+\sigma_H+\sigma'_H\right)}\right]$$

$$+\mathcal{A}_3\left[\frac{(1-v)\tau\omega}{\left(\tau\omega+1-\omega+\sigma_H+\sigma'_H\right)\left(\nu_1+\sigma_H+\sigma'_H\right)}+\frac{(1-v)(1-\omega)}{\left(\tau\omega+1-\omega+\sigma_H+\sigma'_H\right)\left(\nu_2+\sigma_H+\sigma'_H\right)}\right]$$

$$K'_{17}\equiv K_{MH}=\frac{b_1\beta_M\alpha_{MH}}{\sigma_M\left(\beta_M+\sigma_M\right)},K'_{19}\equiv K_{MwH}=\frac{b_2\beta_{Mw}\alpha_{MwH}}{\sigma_M\left(\beta_{Mw}+\sigma_M\right)}$$

where

$$\mathcal{A}_1=\frac{\lambda\alpha_{HH}\beta_H}{(\beta_H+\sigma_H)},\quad \mathcal{A}_2=\frac{b_1\beta_H\alpha_{HM}}{(\beta_H+\sigma_H)},\quad \mathcal{A}_3=\frac{b_2\beta_H\alpha_{HMw}}{(\beta_H+\sigma_H)}$$

The eigenvalues $\bar{\lambda}$, of (8) satisfy the characteristic equation

$$\bar{\lambda}^3-\mathcal{K}_{HH}\bar{\lambda}^2-(\mathcal{K}_{MH}\mathcal{K}_{HM}+\mathcal{K}_{MwH}\mathcal{K}_{HMw})\bar{\lambda}=0 \tag{14}$$

Hence, we see that the control reproduction number is

$$\mathcal{R}_c=\frac{\mathcal{K}_{HH}}{2}+\frac{\sqrt{\mathcal{K}_{HH}^2+4(\mathcal{K}_{MH}\mathcal{K}_{HM}+\mathcal{K}_{MwH}\mathcal{K}_{HMw})}}{2}\qquad\square \tag{15}$$

Note that in the absence of the wolbachia-carrier mosquitoes used for control, we have the basic reproduction number,

$$\mathcal{R}_0=\frac{\mathcal{K}_{HH}}{2}+\frac{\sqrt{\mathcal{K}_{HH}^2+4\mathcal{K}_{MH}\mathcal{K}_{HM}}}{2} \tag{16}$$

The aim of introducing the wolbachia-carrier mosquitoes is essentially to reduce the basic reproduction number to below unity so that the disease will not spread in the population. Note that the difference between $\mathcal{R}_0$ and $\mathcal{R}_c$ is the additional term $4\mathcal{K}_{MwH}\mathcal{K}_{HMw}$ present in $\mathcal{R}_c$. This term does not necessarily increase $\mathcal{R}_0$, rather the presence of the parameters in this term reduces the parameters in the term $\mathcal{K}_{MH}\mathcal{K}_{HM}$, which consequently reduces the basic reproduction number. The six parameters $b_1, b_2, \beta_M, \beta_{Mw}, \alpha_{MH}$ and α_{MwH} are considered more influential in the spread of zika virus disease and its control by *wolbachia*-carrier mosquitoes. This is because in the absence of the wolbachia-carrier mosquitoes, b_1 is large, and the extrinsic incubation period $\frac{1}{\beta_M}$ is small, such that the transmission probability α_{MH} is high. Hence, the basic

reproduction number $\mathcal{R}_0$ is larger than unity, and the disease can spread in the population. On the other hand, after the introduction of *wolbachia*-carrier mosquitoes, b_1 reduces and b_2 becomes large. Also the extrinsic incubation period $\frac{1}{\beta_{Mw}}$ becomes high, hence very low probability of transmission α_{MwH} and the reproduction number reduces to below one. In this case, the infection cannot propagate. Therefore, we choose these parameters to reflect the specifications made above such that $b_1 \ll b_2$, $\frac{1}{\beta_{Mw}} = \frac{2}{\beta_M}$(that is the incubation period is doubled), and $\alpha_{MwH} \ll \alpha_{MH}$. Every other parameter in the model remains the same. Using the values for these 6 parameters and other parameters (see Table 1), we see that $\mathcal{R}_c = 0.1206$, while $\mathcal{R}_0 = 1.1123$. This shows that $\mathcal{R}_0$ has been reduced to less than unity when wolbachia-carrier mosquitoes are introduced. Therefore, zika virus disease cannot spread.

Parameter	*Value*	*Source*	*Parameter*	*Value*	*Source*
Π_H	0.8	assumed	σ_H'	0.001	assumed
μ_H	0.002	assumed	Π_M	0.5	assumed
α_{HM}	0.75	[9]	Π_{Mw}	0.5	assumed
α_{HMw}	0.5	assumed	μ_M	0.81	assumed
δ	0.75	assumed	μ_{Mw}	0.82	assumed
β_H	$\frac{1}{3}$	[15]	κ	0.5	[23]
λ	0.02	[28]	b_1	0.05	assumed
α_{HH}	0.8	[9]	b_2	0.7	assumed
v	0.8	[9]	β_M	1/9	[5]
τ	0.5	assumed	β_{Mw}	1/18	assumed
ω	0.85	assumed	α_{MH}	0.75	assumed
ν_1	0.28	assumed	α_{MwH}	0.001	assumed
ν_2	0.5	assumed	q	0.5	assumed
γ_1	0.5	assumed			
γ_2	0.25	assumed			
σ_H	0.005	assumed			
σ_M	0.15	[23]			

Table 1: Parameter Values used in this model

5 Simulations

Here, we present the result of the model simulations, which is done to further show the efficacy of using this biocontrol method. The matlab in-built *ode*45 is used to solve and simulate the model at the baseline

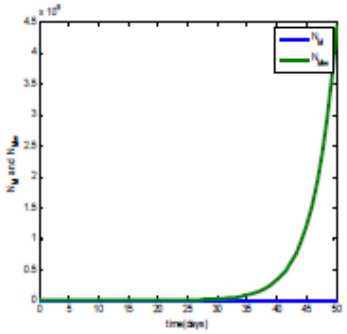

Fig.2: Total Population of Mosquitoes

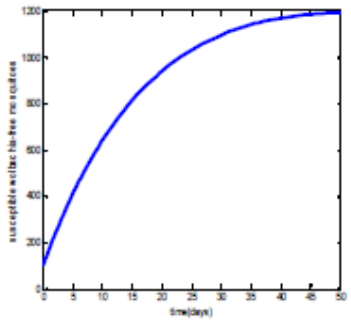

Fig.3: Susceptible wolbachia-free mosquito

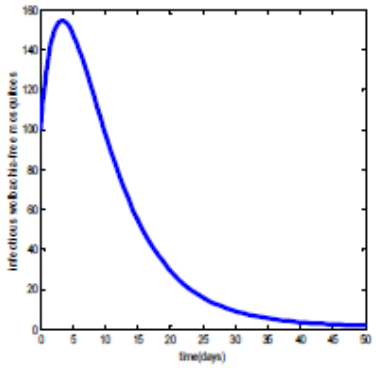

Fig.4:Infectious wolbachia-free mosquitoes

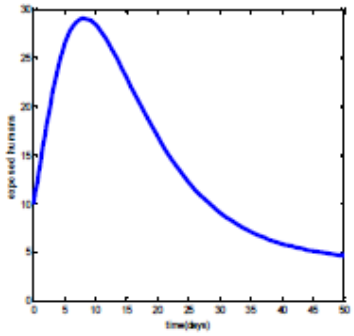

Fig.5: Exposed Humans

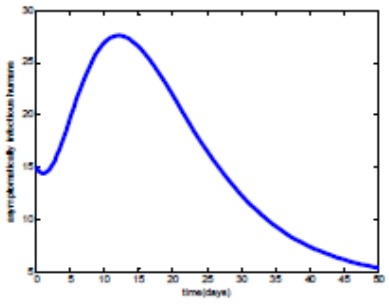

Fig.6: Asymptomatically Infectious humans

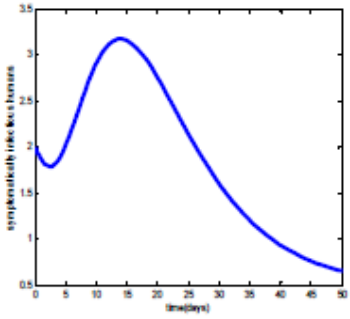

Fig.7: Symptomatically Infectious humans

parameter values(Table 1) with the assumed initial solutions $S_H^0 = 1000, E_H^0 = 10, I_{Has}^0 = 15, I_{Hs}^0 = 2, I_T^0 = 15, I_{NT}^0 = 5, R_{H1}^0 = 0, R_{H2}^0 = 2, S_M^0 = 100, E_M^0 = 500, I_M^0 = 100, S_{Mw}^0 = 1000, E_{Mw}^0 = 0, I_{Mw} = 0$. The result of the simulations is shown in figures (2-7). In figure 2, we see how the wolbachia-carrier mosquitoes have succeeded in displacing the the wolbachia-free ones. Moreover, in figure 3, the susceptible wolbachia-free mosquitoes have been stopped from further increase and in figure 4, the population of infectious wolbachia-free mosquitoes have being reduced to the barest minimum. The resultant effect of the reductions in the population of wolbachia-free mosquitoes on the populations of exposed, asymptomatically infectious humans and symptomatically infectious humans can be easily seen in figure 5, figure 6 and figure 7 respectively. The reduction in the populations of wolbachia-free mosquitoes and infectious human population reduces the reproduction number of the disease to below unity. Therefore, the disease cannot spread.

6 Conclusion

In this work, we have modeled the used of wolbachia-infected aedes aegypti mosquitoes in the fight against zika virus disease. We computed the control reproduction number, $\mathcal{R}_c$, of the disease using the next generation matrix approach. The value of $\mathcal{R}_c$, which is less than one shows that we can stop the spread of zika virus disease by introducing wolbachia-infected mosquitoes in the area where the disease is ravaging

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